



REVIEW ARTICLE

Non-breathing-related sleep disorders following stroke[☆]

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Received 2 October 2012; accepted 21 April 2013

Available online 25 October 2014

KEYWORDS

Cerebrovascular disease;
Stroke;
Sleep;
Sleep disorders;
Parasomnias;
Insomnia

Abstract

Introduction: It has been shown that sleep-related breathing disorders, especially sleep apnoea, are very common in patients who have had a stroke, and that they also reduce the potential for neurological recovery. Nevertheless, other sleep disorders caused by stroke (excessive daytime sleepiness, insomnia, sleep-related movement disorders) can also cause or increase stroke-related disability, and this fact is less commonly known.

Development: Studies with polysomnography have shown many abnormalities in sleep architecture during the acute phase of stroke; these abnormalities have a negative impact on the patient's quality of life although they tend to improve with time. This also happens with other sleep disorders occurring as the result of a stroke (insomnia, narcolepsy, restless legs syndrome, periodic limb movement disorder and REM sleep behaviour disorder), which are nevertheless potentially treatable. In this article, we briefly review the physiopathology and epidemiology of the disorders listed above in order to raise awareness about the importance of these disorders and the effects they elicit in stroke patients.

Conclusions: Sleep disorders that are not breathing-related have scarcely been studied in stroke patients despite the fact that almost all such disorders may present as a result of a cerebrovascular event.

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PALABRAS CLAVE

Enfermedad cerebrovascular;
Ictus;
Sueño;

Trastornos del sueño no respiratorios en relación con ictus

Resumen

Introducción: Actualmente se reconoce que los trastornos respiratorios, en especial la apnea del sueño, son frecuentes en pacientes con accidente vascular cerebral y que su presencia reduce el potencial de recuperación neurológica de estos pacientes.

[☆] Please cite this article as: Marquez-Romero JM, Morales-Ramírez M, Arauz A. Trastornos del sueño no respiratorios en relación con ictus. Neurología. 2014;29:511–516.

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Trastornos del Sueño;
Parasomnias;
Insomnio

Sin embargo, es poco conocido el hecho de que otros trastornos del sueño que también se producen a consecuencia de un ictus como la somnolencia diurna, el insomnio y los trastornos del movimiento también son capaces de producir o incrementar la discapacidad asociada al ictus.

Desarrollo: Estudios polisomnográficos han evidenciado múltiples alteraciones en la arquitectura del sueño de los pacientes en la fase aguda del ictus, las cuales tienden a mejorar con el transcurso del tiempo pero manteniendo un efecto deletéreo sobre la calidad de vida. Lo mismo ocurre con trastornos del sueño que se producen como consecuencia de un ictus (el insomnio, la narcolepsia, el síndrome de piernas inquietas, los movimientos periódicos de las piernas y el trastorno de conducta del sueño MOR) todos los cuales son potencialmente tratables. Con el objetivo de incrementar la conciencia acerca de estas condiciones y sus efectos sobre los pacientes con ictus, se revisa brevemente la epidemiología y fisiopatología en la subpoblación de pacientes neurológicos con ictus.

Conclusiones: A diferencia de los trastornos respiratorios, otros trastornos del sueño han sido escasamente estudiados en pacientes con ictus, a pesar de que prácticamente todos los trastornos del sueño pueden presentarse a consecuencia de esta enfermedad.

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Introduction

Sleep disorders (SD) are present in all age groups and significantly affect patients' quality of life. The second edition of the International Classification of Sleep Disorders (ICSD-2) subdivides these disorders into eight major categories: (1) insomnias; (2) sleep-related breathing disorders (SRBD); (3) hypersomnias; (4) circadian rhythm sleep disorders; (5) parasomnias; (6) sleep-related movement disorders; (7) isolated symptoms, apparently normal variants and unresolved issues; and (8) other sleep disorders.¹

Excellent scientific evidence gathered in the past few years points to an association between SD and cerebrovascular risk/stroke. Therefore, the study of the complex interactions between these two conditions has become an important topic in vascular neurology.²

We currently know that SRBDs, particularly sleep apnoea, are more frequent during the acute phase of a cerebrovascular accident (CVA) and that they reduce the patient's capacity for neurological recovery.³ Additionally, patients with obstructive sleep apnoea experience greater numbers of vascular episodes than healthy subjects do.⁴

However, other SDs may also appear as direct or indirect consequences of stroke.⁵ Aside from SRBDs, insomnia and hypersomnias (excessive daytime sleepiness with fatigue and increased need of sleep) are the most frequent SD subtypes in stroke patients, especially in cases of hemispheric, thalamic, or brainstem infarction.⁶ As with SRBDs, these subtypes can elicit or exacerbate stroke-related disability. Furthermore, although SDs can be identified easily, their presence is usually underestimated and unjustifiably ignored in patients with cerebrovascular disease. These entities are linked to neuropsychiatric disorders and a less favourable functional outcome.⁷

This review aims to summarise international literature addressing the association between stroke characteristics (topography, severity, and outcome) and presence of SDs other than SRBD.

Procedure

Sleep architecture during acute stroke

Normal sleep architecture is severely altered during the acute phase of stroke⁸; multiple factors contribute to this alteration. Firstly, there are factors inherent to the loss of neural tissue (direct lesion to structures linked to the generation or maintenance of the sleep-wake cycle) and subsequent oedema; secondly, we find the consequences of neurological deficit (limited mobility, pain, etc.). The last category describes environmental factors associated with hospitalisation (bed confinement, continuous lighting, noise, etc.).⁶

Sleep quantity is preserved in 52% of the patients during the acute phase of stroke, although total sleep time varies considerably. The number of arousals and hours of daytime sleep increase significantly, and these trends are correlated with poor sleep quality and diminished quality of life.⁹

Polysomnography studies have revealed several alterations in the sleep architecture of patients with acute stroke, such as reductions in total sleep duration and sleep efficiency, increases in sleep latency, and a tendency towards decreased slow wave sleep and rapid eye movement (REM) sleep.^{10,11}

Researchers have also documented a correlation between encephalographic changes and stroke severity as measured by the Scandinavian Stroke Scale (SSS).¹² Another polysomnography study performed by Terzoudi et al.¹³ showed a positive correlation between REM sleep latency and 3-month functional outcome measured by Barthel index. According to their findings, patients with the worst outcomes experienced a marked decrease in REM sleep latency compared with patients with better outcomes. In addition, patients in this study with cerebellar infarctions showed more severely impaired non-REM sleep compared to patients with infarctions in other locations.

In conclusion, polysomnography studies in patients with CVA suggest that normal architecture of nocturnal sleep is

severely altered by the ischaemic insult. This, in turn, elicits changes in the mechanism that regulates sleep architecture and generates and maintains its individual stages.⁸

The generation, organisation, and maintenance of normal sleep architecture all play a very important role in maintaining homeostasis and in the consolidation of learning and memory, including motor skill learning.¹⁴ This last type of learning is essential for recovering motor skills after the acute stage of stroke.¹⁵

The role of normal sleep architecture in the emergence of such comorbidities as depression has also been studied, in both healthy subjects¹⁶ and stroke patients.¹⁷

Lastly, understanding how sleep is affected after a cerebrovascular accident represents an interesting area of research. Current evidence from mouse models supports the neuroprotective effect of sleep,¹⁸ and sleep induction has been suggested as a valid therapeutic strategy to enhance functional recovery of stroke patients.¹⁹

Sleep architecture after acute stroke

Severe disruptions in sleep architecture during the acute phase of stroke seem to normalise once the acute phase has come to an end.²⁰ A study of polysomnography results from 96 patients admitted to a stroke rehabilitation unit²¹ could not identify differences in total time in bed, total sleep time, sleep efficiency, and REM and non-REM sleep quantity between these patients and almost 2000 control subjects with no history of stroke. However, sleep latency remained elevated in patients after CVA. Another study carried out in 20 stroke survivors showed that 45% of these patients had experienced an increase in sleep latency although total sleep duration and efficiency were within normal values.²² No abnormal sleep patterns have been found in patients with acute-phase hemispheric CVA compared to sleep patterns in control subjects.²³ However, a study of patients with subarachnoid haemorrhage (SAH) revealed that 34% of patients reported severe sleep problems. Polysomnography studies of these disturbances detected sleep fragmentation in 75% of the patients, increased sleep latency in 35%, and low sleep efficiency in 55%.²⁴ These findings were correlated to a markedly diminished quality of life.

Hypersomnias of central origin

Excessive daytime sleepiness and pseudo-hypersomnias

When discussing excessive daytime sleepiness (EDS) in patients recovering from CVA, we should differentiate between this syndrome, in which patients who get enough sleep at night display a tendency to sleep during the day (even at inappropriate times such as when eating, driving, or working), and hypersomnia secondary to bilateral paramedian thalamic lesions.^{25,26} This disabling hypersomnia presents concomitantly with other disorders including aboulia, lack of mobility, vertical gaze palsy, memory disorders, and mood swings.^{27–29} In these patients, interruption of waking mechanisms at the level of the reticular formation, thalamic reticular nuclei, or such brainstem nuclei as

the locus coeruleus, reduces the level of consciousness. This reduction is secondary to decreased afferent input from the monoaminergic pathways in the brainstem.^{30,31} Since presence of EDS in patients after a CVA is pathophysiologically different from hypersomnias that accompany vascular syndromes affecting the diencephalon, some authors have coined the term pseudo-hypersomnias³² to refer to these syndromes.

Excessive daytime sleepiness is the most frequent hypersomnia of central origin in stroke patients; in fact, it is the most frequent SD in these patients after SRBD and its role as risk factor for experiencing a CVA has recently been recognised.³³ A study performed in 200 acute stroke patients showed that 49.5% presented at least moderate symptoms of EDS³⁴; its prevalence rate in patients with SAH is 6%, although this percentage might be higher in patients with perimesencephalic SAH.²⁴ In a study by Sterr et al., 40% of patients had severe EDS and EDS scale scores showed a positive correlation with time since CVA. A tendency towards higher prevalence rates for anxiety and depression was also observed, although these patients had normal sleep architecture. This suggests that EDS in patients with CVA may arise independently from any nocturnal sleep disorders.²²

The pathophysiogenesis of EDS after stroke remains unknown, but it could be related to diffuse cortical dysfunction, especially in cases of SAH.²⁴ The above statement is supported by a case of histologically confirmed cortical laminar necrosis in which the polysomnographic study showed absence of slow waves and spikes during sleep (stage N2) and loss of ultradian organisation of sleep.³⁵

Narcolepsy

Narcolepsy, with or without cataplexy, may occur after stroke with a local or diffuse lesion (as in anoxic-ischaemic encephalopathy). In the study by Pasic et al., narcolepsy prevalence in stroke patients was 0.5%³⁴; this prevalence does not differ significantly from that found in the general population (0.047% in Europe³⁶) and its presence in the literature is merely anecdotal. Only 4 cases have been reported as of 2013.^{37–40} The affected anatomical locations were the hypothalamus and rostral mesencephalon⁴¹; these locations are responsible not only for clinical symptoms, but also for a very significant decrease in orexin levels in CSF.³⁷ Lesions in the ventral pons can also elicit clinical symptoms identical to those of idiopathic narcolepsy,³⁸ but these cases do not show decreased orexin levels. Pontine stroke has also been linked to presence of isolated cataplexy, although the lesion in that case was located in the mesopontine region.³⁹ Lastly, we find a case report on a patient with bilateral paramedian thalamic infarctions who experienced recurrent episodes of unresponsiveness; episodes were accompanied by an electroencephalographic pattern similar to that in stage-2 sleep, but there were no other associated clinical manifestations.⁴⁰

Insomnia

Insomnia prevalence rates in patients after cerebral infarction have been reported as 68% during the acute stage⁴² and 49% to 18.1% at 18 months after stroke, when insomnia

frequency increases in line with the degree of disability after CVA.⁴³ Insomnia has been described as a consequence of SAH in 25% to 31% of these stroke patients.²⁴

Acute insomnia has been reported in patients with bilateral or left thalamic ischaemic lesions. Although diagnosis was not corroborated by polysomnography in these cases, the subthalamic lesion, rather than the lesion to the thalamus itself, is thought to explain the presence of insomnia in these patients. This occurs because disruption of the inhibitory connections between the anterior hypothalamus and the rostral part of the reticular formation abolishes the ability to inhibit the waking state, a necessary step in initiating sleep.⁴⁴ Especially in patients with subcortical thalamic, thalamo-mesencephalic, or tegmental pontine CVA, insomnia can be accompanied by an inversion of the sleep-wake cycle which leads to nocturnal insomnia and agitation and daytime hypersomnia.^{45,46} Rapid fluctuations between insomnia and hypersomnia observed in some of these patients emphasise the dual role these structures play in regulating the sleep-wake cycle.⁶ Total insomnia was temporarily present in one patient with lateral bulbar infarction.⁴⁷

Other factors contributing to the development of insomnia in stroke patients include the initial impression of having a severe disorder and the subsequent adaptations to physical or cognitive limitations, depression after CVA, and side effects of drug treatments.⁴⁸

Movement disorders

Although the ICSD-2 lists eight diagnostic categories of sleep-related movement disorders, only two categories are cited in the literature as being linked to CVA: restless leg syndrome (RLS) and periodic limb movements (PLM). RLS is a disorder clinically characterised by a compelling urge to move the limbs (especially the legs) when resting, especially at night; moving the affected limbs produces a sensation of relief. In contrast, PLM describes a condition in which a polysomnography study reveals periodic episodes of four or more repetitive contractions of the tibialis anterior during sleep. Contractions appear every 5-90 seconds and have a duration of 0.5-10 seconds. These contractions cause sleep disorders and daytime fatigue.¹ In 80% to 90% of cases, PLM presents concomitantly with RLS.

Prevalence of PLM/RLS in patients with CVA was 12.4% at one month after stroke according to the only prospective study completed to date.⁴⁹ In the study by Schuiling et al., 25% of the patients with SAH had either RLS or PLM.²⁴

PLM/RLS associated with CVA has mainly been observed in patients with lesions in subcortical structures, such as the basal ganglia,^{50,51} the pons,⁵² and the lateral part of the thalamus,⁵³ listed in order of frequency. Regarding clinical findings, it is important to highlight that no upper limb symptoms were found in any of the stroke patients with RLS/PLM. Symptoms were bilateral in 75% of the cases; in cases of unilateral symptoms, they affected the side contralateral to the lesion.⁴⁹ The above findings seem to support the hypothesis that loss of cortical inhibition of basal ganglia pathways explains the pathophysiology of the disorder, especially ascending disinhibition of the sensorimotor cortex and disinhibition of descending inhibitory

pathways of basal ganglia.⁵⁴ Lastly, we find a case report on a patient with unilateral PLM and no RLS, as confirmed by polysomnography. PLM developed after acute ischaemic stroke in the corona radiata.⁵⁵

Parasomnias

Parasomnias are disorders characterised by abnormal behaviours or physiological phenomena that occur during any of the stages of sleep or sleep-wake transitions. They are caused by the activation of physiological systems (motor, autonomic, or cognitive) at inappropriate moments during the sleep-wake cycle. Parasomnias include nightmares, night terrors, sleepwalking, sleep talking, confusional arousals, bruxism, sleep paralysis, and REM sleep behaviour disorders (RBD).

Prevalence of parasomnias in patients with CVA has not been determined. In the study by Pasic et al.,³⁴ 82.5% of the patients presented PLM/RLS, bruxism, sleepwalking and/or sleep talking, but the percentage for each SD is not provided.

Although total dream loss has been reported after CVA with temporal-occipital damage,^{56,57} no parasomnias except for RBD have been reported in the literature as stroke sequelae. In the case of a patient with apparently recurrent nightmares after a right temporal lobe infarction, researchers concluded that episodes were in fact epileptic seizures since they also took place during wakefulness. Seizures responded to treatment with phenytoin.⁵⁸

RBD is characterised by lack of REM atonia and patients usually report violent dream images. It is the only type of parasomnia documented as a sequel of stroke; there is a case of RBD secondary to a lesion in the pontine segment accompanied by cataplexy with no other symptoms of narcolepsy,³⁹ and two more cases of isolated RBD appearing after paramedian pontine ischaemic lesions.^{59,60} In all cases, damage to the pathways responsible for atonia during REM sleep explains the presence of the disorder, since those pathways involve structures located near the locus coeruleus. They also induce tonic excitatory activity on the magnocellular reticular formation, which inhibits spinal motor neuron activity along the reticulospinal tract during REM sleep.⁶¹

Conclusions

Generally speaking, SDs in adults are associated with symptoms of depression, fatigue, and cognitive deficits.^{62,63} If these disorders are present in patients with CVA, they can interfere with functional recovery by decreasing a patient's energy, motivation, and rest.

To date, researchers have only studied the impact of treating SRBDs on stroke recurrence⁶⁴ and stroke recovery,⁶⁵ but results are promising.⁶⁶ This highlights how important it is to screen for all SDs in patients with CVA. Simple and repeatable instruments, such as the Epworth scale⁶⁷ and the Sleep Disorders Questionnaire,⁶⁸ may be used in daily clinical practice to identify those patients who should undergo polysomnography to confirm or diagnose presence of SD.⁶⁹

Today, resources for identifying and diagnosing patients with SD tend to be available in most countries; numerous

treatment alternatives are also available which may improve quality of life and the recovery potential of stroke patients.⁴⁸ Doctors must therefore assume the responsibility of carrying out these procedures and interventions.

Conflicts of interest

The authors have no conflicts of interest to declare.

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